point of view of the symptoms to which they give rise. The question is not of a more or less deeply penetrating cavity, but of an extensive loss of substance comprising the ventricular mass and leaving only the meninges of the superficial convolutions, as instanced in the right sphenoidal lobe.

Although, according to Kundrat and Audry, the porencephalous cavity is usually single, it is sometimes double, oftenest symmetrically placed on the lateral hemispheres; and in proportion of 38 to 26 it occurs on the left, as in the present case. Porencephalitis usually develops in intrauterine life, rarely in early years. The patient under consideration was an exception, as the lesions were developed later, according to his history; the bony protuberance in the left sphenoidal fossa, where the alteration was most distinct, was also proof that the accident was of long standing but not of infancy, which would have caused injury of another sort. He had probably lived twenty-six years after the accident, and had grown better instead of worse, as is the case in porencephalitis. The fact that general sensibility and muscular control,as well as the senses of taste and smell, were normal in the patient, proves that the centers of their various manifestations must be elsewhere than in the hippocampal region, in the Rolandic, for instance, contrary to the suppositions of most physiologists.

3. *Cerebellar Hemiasynergie with Autopsy.—Hematic cyst in the right cerebellar hemisphere.*—The cyst was the remains of a former hemorrhage, found at autopsy fastened in the white substance of the right hemisphere, to which was added, as the immediate cause of death, a profuse hemorrhage retained in the cavity. The anatomo-clinical methods employed justify the following deductions. (1) The value of hemiasynergia for diagnosis of protuberant cerebellar lesions of the same side was first shown by Babinski. By this sign, in the actual case, the cause of the cerebellar syndrome was localized in the right side, and this topographical diagnosis was confirmed by the autopsy. Microscopical examination alone will ultimately determine whether the recent protuberant lesion does not hide an older lesion. Moreover, even in this very case, the anatomo-clinical observation, even if it does not show the cerebellar nature of the hemiasynergia, always shows its protuberant nature. (2) The existence of paradoxical reflexes before pain began and seemingly allied to the recent hemorrhage. This is a fact which must wait for confirmation, and the clinical value of which entirely escapes us.

JELLIFFE.

Revue Neurologique.

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1. Flaccid Paraplegia in a Case of Cervical Pachymeningitis. E. BRISSAUD AND MAURICE BRÉCY.
2. Remarks on Permanent Spasmodic Paraplegia through Medullary Tumor. RAYMOND AND R. CESTAN.

1. *Flaccid Paraplegia.*—There is brought forward a new fact concerning paraplegia, observed in the course of a tuberculous cervical pachymeningitis. There has been formerly presented a case of spasmodic paraplegia produced by compression of the dorsal medulla, equivalent to a section. The infallibility attributed to the law of Bastian by most neurologists is not recognized for the following reasons: "We do not dispute that complete and sudden section of the medulla, which produces certain traumatisms, induces paraplegia, always flaccid, with no reflexes and total loss of sensibility; but until more fully instructed, we consider as proved the fact that a slow pressure acting as a ligature with

an indefinitely prolonged structure, transforming the tissue of the medulla into a veritable cicatrix, may give place to spasmodic paraplegia."

The present case seems to contradict this. A tuberculous patient who showed signs of cervical pachymeningitis for the first time in April, became paraplegic in May. The paraplegia was a flaccid one and he died in September, five months thus passing between symptoms and death. Often apparently merely descending lesions of the lateral cords, either by cutting off inhibitory action of the brain or by emphasizing the excitomotor power of the anterior cornua, regulate the spasmodic condition of cases of progressive paraplegia. In fact, the question is of sudden or apoplectic paraplegia. An hypothesis of the pathogeny of such flaccid paraplegias, which seems at least logical, is ventured: The myoneure is excited from two principal sources, the pyramidal neuron and the centripetal protoneuron, of equal importance, as the suppression of one is fatal to the reflex.

The history is as follows: A man of twenty-five was admitted to the hospital on the first of April with a cold abscess on the upper internal part of the right thigh, situated immediately above the perineo-crural fold. The diagnosis was osteitis of the ascending branch of the ischium. After cocainization of the spine the abscess was lanced and for several days the wound continued to discharge pus. On the 24th the pain was felt in the right shoulder and scapula and there was difficulty in extending the fingers. May 4, there was a slight diminution of the faradic and galvanic excitability of the triceps, radialis, posterior ulna and finger extensors, also the adductor of the thumb. The supinator longus was intact, on account of which fact a vain search was made for lead poisoning. By the middle of May the scapular pains had extended throughout the thorax and the upper limbs; the pain was spontaneous, continuous and severe, unaffected by manipulation. Movement was possible in the left hand, but muscular force was diminished; in the right were all symptoms of radial paralysis of central origin; the arm being raised, the forearm fell; hand extension was very difficult; lateral movements and those of fingers and phalanges were impossible. Already there was slight atrophy of the fore-arm and of the thenar and hypothenar surfaces. The electric symptoms were emphasized, but the supinator longus was still normal. Dimution in galvanic excitability was parallel to the faradic. Thus twenty-two days after first scapular pains there was paralysis of the radial nerve with muscular atrophy, except for the supinator longus. The lower limbs were unaffected though the wound of the thigh continued to discharged pus. Twenty-seven days after the first symptoms there was some weakness of the lower limbs, and the next day there was complete paraplegia, with fever. Both legs were impotent, although there were patellar reflexes, exaggerated on the right; plantar reflexes were weak but normal. Sensibility to touch was present, but not to pain, heat or cold; there was retention of urine. Presently the sense troubles extended to the umbilicus and lumbar puncture brought a slightly rose-colored liquid. Fever persisted, the tongue was dry, breath rapid; there was pleurisy on the right with a slight discharge; an eschar appeared on the left buttock and another on the right external malleolus. There was retention of urine and incontinence of fecal matter. One eschar ulcerated and a third appeared on the left malleolus, a fourth on the left heel. The entire left leg was edematous.

Anesthesia extended to the base of the thorax, and was absolute for all senses, stopping at a hypothetical inframammary zone. Plantar and tendinous reflexes were gone, only that of the fascia lata remaining. Hard pinching of the legs caused reflex movements of flexion, but pain was not perceived. A slight ptosis of the right eye next appeared, with myosis and exophthalmia, without diplopia. Movements of the ball were normal. Deglutition was slightly painful. Edema extended to both legs. August 8, there was broncho-pneumonia in the right, and signs of tuberculous pulmonary infiltration. The general state grew gradually worse, with noctural delirium, oppression, polypnea, cyanosis. Death occurred September 4.

Autopsy.—Pulmonary tuberculosis, tuberculous osteitis of the right ischiopubic branch. Cranial meninges unaffected. External tuberculous pachymeningitis in the cervicodorsal region, extending from the third cervical to the first dorsal exclusively. A sheath of mamillated fungoid tissue, almost 1 cm. thick, envelopes this part of the spinal cord. In all this region the anterior and posterior roots are imbedded in fungoid masses, and entagled with the mass, the parts of which were recognizable only with the microscope. The microscopic alterations of the nervous substance were many, both in white and gray matter. It is especially to be noted that a large number of conductors are not involved in the anterior columns and in the lower lateral zones.

Compression of the cord was not the cause of this interruption of nervous tracts in white and gray matter; but acute inflammation of a very limited portion of the axis.

2. *Spasmodic Paraplegia and Medullary Tumor.*—What result is brought to the tendinous reflexes of the lower limbs by total transverse destruction of a dorsal or cervical segment of the spinal cord? There are two solutions given in this paper by Raymond and Cestan. According to certain neurologists the patellar reflex, to be evident, needs integrity of the medullary lumbar arc formed by the sensory ganglionic neurone and the motor neurone of the corresponding anterior cornu. If this intact reflex arc is separated from the encephalon by complete section of the dorsal or cervical medulla, the patellar reflex will disappear through shock to the lumbar region, but will soon reappear and there will be a slow, spasmodic paraplegia. If the paralysis of the flaccid type preserves its flaccidity, to explain the absence of contractions, one ought to section the lumbar segment, the medullary roots or the peripheral nerves. According to this opinion the lumbar cord possesses a veritable autonomy and the tracks of tendinous reflexes would be short ones, not passing to the encephalon. In opposition to this medullary theory is one which brings into play the encephalic centers; according to it a tendinous reflex cannot do merely with the intact sensory-motor lumbar arc, unless it is also in relation with certain superior encephalic centers. It is then simply by the mechanism of separation of the lumbar center from the encephalic centers that a complete and sharp section of the medulla determines a flaccid paralysis in spite of the appearance of descending sclerosis of the pyramidal fasciculus.

Often authors have considered only short, or at least subacute sections of the medulla, resulting in fracture of the vertebral column, vertebral osteitis which gives way, pachymeningitis, meningo-myelitis, or in very rapidly progressing tumor. Numerous facts have plainly shown that rapidly-appearing lesions accompany, in case of complete section, flaccid paraplegia, whatever hypothesis be taken to account for the flaccidity. Is the case similar with slow and progressive section? If by chance even a very slow evolution by an infectious meningomyelitis accompanies the transformation of a spasmodic into a flaccid paraplegia, one would suppose, from the toxic and infectious nature, that meningomyelitis would alter the lumbar neurones, whose integrity is indispensable to the manifestation of tendinous reflexes. The ideal process would thus be transverse medullary destruction of long duration, progressive, made day by day and by a mechanical process without the intervention of a toxic-infectious factor. The experimental conditions can not be realized, especially in the ape, whose tendinous reflexes are nearest to those of man. Thus experimental physiology cannot solve the problem, but one must resort to the anatomo-clinical method. There exists in pathology a category of medullary tumors, psammoma, well localized, slow of development, which, starting on the meninges, little by little include the medullary segment in the fashion of a hard, foreign body, whose increase finally determines the destruction of the segment. A veritable experience of pathological physiology accompanies these. Two cases were observed of this sort, both of whom for several years presented a paraplegia always spasmodic with spinal trepidation, to the moment of death; at the end there was added anesthesia of the lower limbs, incontinence of urine and eschars. At autopsy it was found that a psammoma had filled the medulla in the region of the eighth dorsal segment. It may be objected that in spite of evidences the cord was not completely destroyed in the locality of the compression. Although a few nerves were found, these were included by the tumor and moreover complete degeneration of all fasciculi was observed above and below the impression. Granulations were also seen in the zones where the posterior and lumbar roots entered, and in spite of this, which would have involved flaccidity and lent weight to the point of view of destruction of the seventh dorsal, paraplegia was always markedly spasmodic, the patient's legs were rigidly extended and very active in the tendon reflex.

To explain the persistence of spasmodic paraplegia, there are but two hypotheses. It may be supposed that the tendinous reflexes, in adult as in the child, travel simply by the short medullary tracks and that the encephalic centers do not intervene in the production of tendinous reflexes and contraction. Hence one adopts the theory of the abnormal excitability of the short reflex track uniting the posterior cornu to the anterior by the sclerotic tissue of the pyramidal fasciculus; or the theory of the suppression of the inhibitory function of the pyramidal fasciculus; consequently in the cases, destruction of the dorsal medulla with secondary degeneration of the pyramidal fasciculus caused a spasmodic paraplegia. JELLIFFE.

PERISCOPE.

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1. The Trial, Execution, Autopsy and Mental Status of Leon F. Czolgosz, alias Fred. Nieman, the Assassin of President McKinley. CARLOS F. MACDONALD.
2. The Post-mortem Examination of Leon F. Czolgosz, the Assassin of President McKinley. EDWARD ANTHONY SPITZKA.
3. The Twentieth Century Methods of Provision for the Insane. FREDERICK PETERSON. Discussion. CARLOS F. MACDONALD.
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8. Traumatic Encephalitis. HENRY P. FROST.
9. A Review of the Pathological Work Done in the Hospital for the Insane at Independence, Iowa. GERSHOM H. HILL.
10. The Pathology of Insanity. LOUIS C. PETIT.
11. A Case of Idiopathic Internal Unilateral Hydrocephalus with Recurrent Hemiplegic Attacks. WILLIAM CHARLES WHITE.